LETTER TO THE EDITOR

Transient Locked-in Syndrome after Aneurysmal Subarachnoid Bleeding due to Spasm Hypoxemia?

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Dear Editor,

We read with interest the article by Vaithialingam et al. about a 60-year-old female with a history of arterial hypertension who experienced subarachnoid bleeding (SAB) and intraventricular bleeding due to an aneurysm of the intracranial portion of the internal carotid artery.[1] The patient was comatose with dilated pupils upon admission, received external ventricular drainage, and was discharged at the request of her relatives.[1] Upon readmission 2 days later, the patient responded only with eye blinking and vertical bulb movements, was diagnosed with locked-in syndrome (LIS), and underwent successful coiling of the aneurysm.[1] Three days after coiling, slight movements were observed, and she continued to regain at least some of her motor functions until the last follow-up.[1] The study is impressive, but several points require discussion.

Why was no computed tomography angiography (CTA) performed upon admission to the first hospital? In patients with a SAB, it is imperative to perform CTA at diagnosis to determine whether the SAB was due to rupture of an aneurysm or other nonaneurysmatic causes, such as trauma, dural fistula, arteriovenous malformation, central nervous system (CNS) vasculitis, carotid artery dissection, intracranial artery dissection, or shaken baby syndrome.

It is unclear why cerebrospinal fluid (CSF) analysis was reported as normal.[1] In a patient with SAB and ventriculostomy several days prior, CSF examination is expected to reveal elevated white blood cells, elevated erythrocytes, elevated hemoglobin, elevated bilirubin, high sodium, and elevated protein.[2] The discrepancy between the index case and what has been reported in the literature[2] should be explained.

Subarachnoid bleeding is often complicated by spasms of intracranial arteries, often complicated by ischemic stroke.[3] Was there any evidence for arterial spasms, particularly of the basilar artery, during follow-up CTA? Is it conceivable that the LIS was due to hypoxemia due to spasms of the penetrating branches of the basilar artery that resolved spontaneously over time?

Why was the patient discharged to home care and did not undergo neurosurgical evaluation immediately upon admission? It is incomprehensible why treating physicians allowed the patient to be discharged with an external ventricular drain. Was the external ventricular drain removed before discharge, or was the patient discharged with the drain in place?

We should know what modalities were used for MRI. Was there DWI hyperintensity or cytotoxic edema in the brainstem?

In conclusion, the excellent study has limitations that should be addressed before drawing final conclusions. Clarifying the weaknesses would strengthen the conclusions and could improve the study.

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Author Contribution

JF was responsible for the design and conception, discussed available data with co-authors, wrote the first draft, and gave final approval.

Availability of Data and Material

All data are available from the corresponding author.

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